Precision Natinal Corporation (PNC) is a machine shop that repairs large crankshafts, large ID and OD rods, and cylinders by chrome plating and grinding. The primary wastes produced by PNC are:

1) grinding and polishing waste

2) Chromate Bearing waste (Hexaralent Cr)

3) Chromate Bearing sludge

They have been producing, and disposing of wastes since 1962.

Prior to 1977, PNC reportedly dumped approximately 40 gallons of coolant mixed with a maximum of 1/2 cubic yard of metal grindings per month in a field just behind the plant, per a correspondence memo between TWQB and plant manager Mr. William P. Matthews.

Since 1977, waste have been sent through a treatment process operated by the plant.

Mr. Matthews believes that the elevated levels of  ${\rm Cr}^{6+}$  and  ${\rm Cr}^{3+}$  found in the recon inspection were caused by past dumping practices and are not due to spillage and migration from the treatment process area.

Analysis of thirteen (13) soil sample collected by FIT on June 20, 1982, revealed that elevated levels of  ${\rm Cr}^{3+}$ ,  ${\rm Cr}^{6+}$ , Pb and other inorganics do exist on site and that some are migrating offsite towards a residental area. Some significant results:

Cr<sup>3+</sup> 3080 ppm 2450 ppm 1100 ppm 1030 ppm 618 ppm

Approx. average background ≤ 10 ppm

Cr<sup>6+</sup> 180 ppm 36 ppm 35 ppm

Approx. average background  $\leq$  5 ppm

Pb 2020 ppm 105 ppm 91 ppm

Approx. average background  $\leq$  15 ppm



Some of the elevated levels of  ${\rm Cr}^{3+}$ ,  ${\rm Cr}^{6+}$  and Pb were found near an area of suspected dumping. Elevated levels of Cr were also found near the treatment processing area. This may indicate that Cr is escaping containment.  ${\rm Cr}^{6+}$  was not detected offsite, however,  ${\rm Cr}^{3+}$  was found at 618 ppm.

The plant is located in a businesss district, however, it is bordered by a Railroad track which is bordered by a residential area.

The site is reportedly not in a recharge. The closest surface drinking water supply is approximately 4 miles away. There are no known drinking water wells within 1/4 miles radius of the site.

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hazardous. Diborane is an irritant to the lungs and kidneys. Decaborane and pentaborane are central nervous system poisons; however, the liver and kidneys may also be damaged if the exposure is severe (Browning, 1969).

## **CESIUM**

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arll st Occurrence and Use. Cesium occurs in nature as pollucite, a hydrous cesium-aluminum silicate. Its main industrial uses are as a catalyst in the polymerization of resin-forming materials and in photoelectric cells. It is useful in this respect because the range of sensitivity is approximately that of the human eye. Radioactive cesium is a constituent of nuclear fallout.

Absorption, Excretion, Toxicity. Cesium is absorbed after oral administration and is bound within the cells of the soft tissues such as kidney and muscle. It is found in the red blood cells and may in some circumstances be able to replace potassium. The urine is the main route of excretion. Increased potassium levels facilitate cesium excretion. The radioactive material is found in milk.

No cases of industrial injury related to the chemical toxicity of cesium have been reported. It is likely that replacement of potassium by cesium would produce ill effects in man, probably neuromuscular in nature, as has been demonstrated in experimental animals (Browning, 1969).

## CHROMIUM

Occurrence and Use. Chromite (FeCr<sub>2</sub>O<sub>4</sub>) is the most important chrome ore. Chromium plating is one of the major uses of this metal. Steelfabrication, paint and pigment manufacturing, and leather taning constitute other major uses of chromium. The medicinal uses of chromium are limited to external application of chromium trioxide as a caustic and intravenous sodium radiochromate to evaluate the life-span of red cells.

Absorption, Excretion, Toxicity. Chromium exists in several valence states. Only the trivalent and hexavalent are biologically significant. While conversion from trivalent to hexavalent and other states is important chemically, the inner conversion from chromic to chromate does not apparently occur biologically. The conversion of hexavalent to trivalent does take place in the body.

Trivalent chromium is an essential element in animals. It plays a role in glucose and lipid metabolism. Chromium deficiency mimics diabetes mellitus and produces aortic plaques in rats. Chromium supplementation improves or normalizes glucose tolerance in diabetics, older people, and malnourished children. It has been suggested that chromium deficiency may be a basic factor in atherosclerosis (Mertz, 1969; Schroeder et al., 1970c). A deficiency of trivalent chromium apparently increases the toxicity of lead (Schroeder et al., 1965).

The major environmental exposure to chromium occurs as a consequence of its presence in food. Brown sugar and animal fats, especially butter, are chromium-rich foods. Chromium is found in urban air (Table 17-3). The concentration in natural water supplies is below 10 ppb; however, in municipal drinking water concentrations of 35 ppb have been reported (Table 17-2). The daily intake has been estimated at  $60 \mu g$  (30 to  $100 \mu g$ ),  $10 \mu g$  of which is due to water concentrations (Table 17-1). However, the absorption is limited to approximately 1 percent (Schroeder et al., 1962b). The occurrence of chromium in food or water has not been shown to produce any significant adverse effects in either man or experimental animals (U.S. Public Health Service, 1962; Kanisawa and Schroeder. 1969; Schroeder and Mitchener, 1971).

The total chromium body burden of man has been estimated at less than 6 mg (Table 17-1). Chromium is transported across the placenta and concentrated in the fetus. The tissue concentrations tend to decline rapidly with age except for the lung concentration, which tends to increase. The decline of chromium levels with age does not occur in rats. Wide geographic variations in tissue concentration, presumably due to differences in dietary intake and atmospheric concentration, have been reported (Schroeder et al., 1970d).

Water-soluble chromates disappear from the lungs into the circulatory system after intratracheal application, while the trivalent chromic chloride remains largely in the lungs. Oral administration of trivalent chromium results in little chromium absorption. The degree of absorption is slightly higher following administration of hexavalent compounds. Once absorbed, Cr<sup>3+</sup> is bound to the plasma proteins. Under normal conditions the body contains stores of chromium in the skin, lungs, muscle, and fat. The bone contains chromium, but this is not due to selective deposition. The caudate nucleus has been reported to have high concentrations. Hexavalent chromium is reduced to the trivalent form in the skin. In the blood little hexavalent chromium can be detected. The reticuloendothelial system, liver, spleen, testes, and bone marrow have an affinity for chromite, possibly as the result of phagocytosis of colloidal particles formed at higher tissue concentrations. On the other hand, chromates are bound largely to the red blood cells. Subcellular distribution studies have indicated that the nuclear fraction

contains almost one-half the intracellular chromium. Urinary excretion accounts for about 80 percent of injected chromium. However, elimination via the intestine may also play a role in chromium excretion. Milk is another secondary route of excretion (Mertz, 1969). Average urinary and blood concentrations are 0.4 and 2.8  $\mu$ g/100 g, respectively (Imbus et al., 1963).

Occupational exposure to chromium compounds (Cr6+) causes dermatitis, penetrating ulcers on the hands and forearms, perforation of the nasal septum, and inflammation of the larynx and liver. The dermatitis is probably due to an allergenic response, although persons sensitive to Cr6+ also respond to large amounts of Cr3+ (Fregert and Rossman, 1964). The ulcers are believed to be due to chromate ion and not related to sensitization. Chromic acid, and, to a lesser extent, chromate, are presumably the causative agents in perforation of the nasal septum (Browning, 1969). Epidemiologic studies indicate that chromate is a carcinogen with bronchogenic carcinoma as the principal lesion. The latent period appears to be 10 to 15 years. The relative risk of chromate plant workers for respiratory cancer is 20 times greater than that of the general population. Experimental studies have suggested that calcium chromate may be the specific carcinogenic agent (Enterline, 1974). However, some investigators have produced cancer in experimental animals with injections of either the trivalent or hexavalent form (Hueper and Payne, 1962). Incorporation of hexavalent chromium (5 ppm) into the drinking water of mice over their lifetimes produced a slightly higher incidence of malignant tumors than in the controls. Trivalent chromium (chromium acetate) given to rats under similar conditions produced no such effect (Schroeder and Mitchner, 1971; Kanisawa and Schroeder, 1969).

## COBALT

Occurrence and Use. Cobalt is a relatively rare metal produced primarily as a by-product of other metals, chiefly copper. It is used in high-temperature alloys and in permanent magnets. Its salts are useful in paint driers, as catalysts, and in the production of numerous pigments. It is an essential element in that  $1 \mu g$  of vitamin  $B_{12}$  contains 0.0434  $\mu g$  of cobalt. Vitamin  $B_{12}$  is essential in the prevention of pernicious anemia. If other requirements exist, they are not well understood. Deficiency diseases of cattle and sheep caused by insufficient natural levels of cobalt are characterized by anemia and loss of weight or retarded growth.

Absorption, Excretion, Toxicity. Cobalt salts are generally well absorbed after oral ingestion,

probably in the jejunum. Despite this fact, increased levels tend not to cause significant accumulation. About 80 percent of the ingested cobalt is excreted in the urine. Of the remaining, about 15 percent is excreted in the feces by an enterohepatic pathway, while the milk and sweat are other secondary routes of excretion. The total body burden has been estimated as 1.1 mg.

The muscle contains the largest total fraction, but the fat has the highest concentration. The liver, heart, and hair have significantly higher concentrations than other organs, but the concentration in these organs is relatively low. The normal levels in human urine and blood are about 98 and 0.18  $\mu$ g/1, respectively. The blood level is largely in association with the red cells.

Significant species differences have been observed in the excretion of radiocobalt. In rats and cattle 80 percent is eliminated in the feces (Schroeder et al., 1967b).

Polycythemia is the characteristic response of most mammals, including man, to ingestion of excessive amounts of cobalt. Toxicity resulting from overzealous therapeutic administration has been reported to produce vomiting, diarrhea, and a sensation of warmth. Intravenous administration leads to flushing of the face, increased blood pressure, slowed respiration, giddiness, tinnitus, and deafness due to nerve damage (Browning, 1969).

High levels of chronic oral administration may result in the production of goiter. Epidemiologic studies suggest that the incidence of goiter is higher in regions containing increased levels of cobalt in the water and soil (Wills, 1966). The goitrogenic effect has been elicited by the oral administration of 3 to 4 mg/kg to children in the course of sickle cell anemia therapy (Browning, 1969).

Cardiomyopathy has been caused by excessive intake of cobalt, particularly in beer to which cobalt was added to enhance its foaming qualities. The onset of the poisoning occurred about one month after cobalt was added in concentrations of 1 ppm. Why such a low concentration should produce this effect in the absence of any similar change when cobalt is used therapeutically is unknown. The signs and symptoms were those of congestive heart failure. Autopsy findings revealed a tenfold increase in the cardiac levels of cobalt. Alcohol may have served to potentiate the effect of the cobalt (Morin and Daniel, 1967).

Hyperglycemia due to alpha cell pancreatic damage has been reported after injection into rats. Reduction of blood pressure has also been observed in rats after injection and has led to some experimental use in man (Schroeder et al., 1967b).

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